August 5, 2016

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Subject: Expert Report of Robert J. Myerburg, MD in the Matter of the Death of Andrew Layton - *Cheri Hanson, As Trustee For The Next Of Kin Of Andrew Layton v Best et. al.;* United States District Court, District Of Minnesota, File Number: 15-cv-4578 (MJD/SER)

Dear Mr. Behrenbrinker:

I have reviewed materials you provided to me on the case cited above, and I am submitting herewith my report on the Gold Cross Ambulance paramedic response to, and most probable cause of, the cardiac arrest of Mr. Andrew Layton on January 1, 2013 and his consequent death on January 5, 2013. For preparing this report, I have reviewed and considered the following materials:

- Complaint
- Gold Cross Ambulance Patient Care Reports (Run #69 and Run #70)
- AED (defibrillator) printouts from January 1, 2013
- Records from Mankato Hospital - 01/01/2013 to 01/05/2013
- All medical records of Andrew Layton from Mankato Clinic,
- Medical records of Andrew Layton from the Mayo Clinic Health System - Mankato Hospital for service on 10/19/2013
- Imaging CDs from the Mayo Clinic facility from 10/19/2012 to 01/05/2013
- Ramsey County Medical Examiner's Autopsy Reports;
- Autopsy microscopic slides;
- Deposition transcript of Dr. Butch Huston, MD, Ramsey County Medical Examiner
- Mankato Police Officer Reports and Supplemental Reports
- Medical records for Andrew U.S. Army Reserve - MN National Guard, starting in April 2006;
- Time line prepared using times listed in various videos provided
- CD containing autopsy photographs
- Deposition transcript of Michael Burt, paramedic

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- Deposition transcript of Thomas Drews, paramedic
- Deposition transcript of Dr. Butch Hanson, forensic pathologist
- Deposition transcript of Officer Kenneth Baker
- Deposition transcript of Officer Daniel Best

In formulating my opinions in regard to this case, I have relied upon my educational background, knowledge, and experience in the diagnosis and treatment of cardiovascular disorders generally and of cardiac arrest in particular. I also applied my knowledge and experience regarding the causes of and circumstances surrounding cardiac arrest and its consequences, specifically neurological, metabolic, and respiratory complications.

All of the opinions and conclusions set forth in my report are made to a reasonable degree of medical probability and I have carefully set forth the physical, physiological, anatomical or other scientific basis for each such opinion or conclusion.

I. Background and Qualifications.

I hold the rank of Professor in the Departments of Medicine and Physiology, and the American Heart Association Chair in Cardiovascular Research, at the University of Miami Miller School of Medicine. For the 31 years, from September 1, 1973 through August 31, 2004, I served as Director of the Division of Cardiology at the University of Miami School of Medicine, and Chief of the Cardiology Service at Jackson Memorial Hospital. Since stepping down from the position of Division Chief on September 1, 2004, I have continued as a full-time faculty member at the University of Miami, with duties that include patient care, teaching, and research. I also continue to be directly involved in the cardiovascular medicine and cardiac electrophysiology training programs at the University of Miami.

I am certified by the American Board of Internal Medicine in the fields of Internal Medicine and Cardiovascular Medicine, and have held a time-limited Certificate of Added Qualifications in Clinical Cardiac Electrophysiology (CCEP). The latter was awarded as a consequence of my serving three consecutive 2-year terms on the writing committee for the CCEP board qualifying examination and one term as chair of the self-evaluation examination writing committee, both being components of the American Board of Internal Medicine (ABIM) testing and certification system. I am trained in both Basic Life Support (BLS) and Advanced Cardiac Life Support (ACLS), and am currently an American Heart Association Certified Basic Life Support (BLS) provider.

In addition to an active involvement in clinical practice, teaching and research at the University of Miami, I continue to lecture elsewhere, including national and international scientific meetings, and have served as a consultant for various

academic and commercial elements in the cardiovascular enterprise. I recently served on a committee of the Institute of Medicine of the National Academy of Science, Engineering, and Medicine, convened to evaluate the current status of responses to cardiac arrest in the United States and recommendations for actions that will result in improvements in outcomes.

During the course of my career, I have remained active in clinical practice and teaching in all aspects of cardiovascular medicine, including coronary artery disease and its consequences, heart muscle diseases due to causes other than coronary artery disease, diseases of the heart valves, and infectious and inflammatory diseases of the heart, including the valves, muscle, and pericardium. Throughout my career, I have maintained a particular interest in cardiac arrhythmias, cardiac arrest, and sudden cardiac death, the various causes that result in these conditions, and their clinical consequences.

Among my specific areas of interest, I have been directly involved in research and clinical activities regarding patients at risk for heart attacks and sudden cardiac arrest, as well as research into the clinical use, guideline development, and decision-making strategies for implantable cardioverter-defibrillators (ICD's).[1-4] During the past 15 years, I have also become actively involved in cardiovascular genetics as Director of a Cardiovascular Genetics Clinic in the Division of Cardiology at the University of Miami. My interest in genetics focuses on inherited arrhythmia risk and the genetic epidemiology of sudden cardiac death.[5,6] These various fields of interest require me to be thoroughly familiar with the conditions that cause cardiac arrest, the expected responses to cardiac arrest by physicians, paramedics and emergency medical technicians, trained lay responders, and lay bystanders, and the expected outcomes in victims.

II. Overview of Sudden Cardiac Arrest

Sudden cardiac arrest (SCA) is defined as an abrupt and unexpected loss of consciousness due to a sudden loss of cardiac function in a person with or without known preexisting heart disease.[3] According to generally applied definitions for most spontaneous clinical circumstances, it occurs within one hour after an abrupt change in the victim's clinical status. For circumstances in which cardiac arrest is associated with or triggered by physical or stress-related circumstances, the timing of onset is determined by the specific cause. The consequences of SCA are measured in terms of 3 potential outcomes:

1. Survival free of neurological injury;

2. Survival with persistent neurological injury, ranging from impaired ability to performs various activities of daily living to a permanent vegetative state;

3. Death attributable to the SCA within 30 days, in which case it is referred to as sudden cardiac death (SCD).

The statistics for these categories of outcomes are determined by the integration of multiple factors, the most important of which are the time between onset of cardiac arrest and return of spontaneous circulation (ROSC), effective bystander CPR given prior to ROSC, the electrical mechanism of the SCA (see below – Section III), and the nature and severity of underlying heart disease.[1] The overall survival currently is approximately 10%, driven largely by the fact that up to 80% of SCA's occur in the home, which portends a survival rate of only 6%. Witnessed SCA in public locations has better outcomes, especially when EMTs, paramedics, or lay responders are present at the time of onset and have a portable defibrillator (automated external defibrillator – AED) available. By way of example, the highest reported survival rates come from the Las Vegas casinos, where security guards have AED's immediately available, and survival of those with shockable rhythms is 70% if responded to within 3 minutes.

More than 350,000 out-of-hospital SCAs causing SCD are estimated to occur in the United States each year,[4] representing an annual incidence of 1 SCD per 1,000 persons in the general population over the age of 35 years. The incidence can be considerably higher in subgroups of the population selected for the presence of advanced heart disease. The cumulative figure of 350,000 or more constitutes approximately 50% of all cardiac deaths in the United States, and 15-20% of all natural deaths. However, a different set of statistics apply to younger subsets of the population, especially those from the mid-teen years up to the age of 30 years, among whom the risk of SCA is extremely low. In this subpopulation, the annual risk of naturally-occurring SCD is 1 SCD per 100,000 persons, or 0.001%. This figure is 1% of the risk in the middle-aged and older adult population.

In most instances, SCA occurs as a direct consequence of an abrupt change in the electrical rhythm of the heart. In the past, the most common electrical disturbance (defined as an arrhythmia) responsible for SCD was ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT), which had been estimated to be the primary electrical disturbance in 65-80% of the deaths. Recent data, however, indicates that a lower percentage of out-of-hospital cardiac arrests, much less than 50%, are associated with VF or VT as the initially observed rhythm.[1,3]

The other commonly associated electrical mechanisms are those referred to as non-shockable arrhythmias, which include asystole (complete cessation of electrical activity of the heart – electrical quiescence), bradyarrhythmias (extreme slowing of the heart rate), and pulseless electrical activity (PEA), in which there is electrical activity that does not generate a mechanical heartbeat. The effect of each of these mechanisms, including VF, is the inability of the heart to pump blood; and therefore, there is no blood supply to the other organs of the body, including the brain.

Asystole and PEA occur in a number of different circumstances. In contrast to VF and pulseless VT, PEA and asystole have always been more common mechanisms of cardiac arrest when it is secondary to breathing problems, such as restraint of breathing, asphyxia, or pulmonary embolism, or when it is due to severe blood loss, or to shock due to overwhelming infections. These mechanisms also occur as the primary mechanism in patients with end-stage heart disease, or other disorders such end-stage cancer, or after failure to revert VT or VF because of prolonged cardiac arrest.

All forms of heart disease can be responsible for SCD. Generally, the fact that 50% of all heart-related deaths are sudden applies individually to each of the specific heart diseases. In a few conditions, the proportion of sudden deaths is higher than 50%. The most common cause of SCD in adults in Western civilizations is atherosclerotic heart disease (coronary artery disease) due to cholesterol deposits in the coronary arteries and resulting heart attacks that trigger VF. This one cause accounts for approximately 80% of all SCDs. The second most common cause is a group of disorders called the cardiomyopathies, which collectively share the feature of deterioration and/or dysfunction of the heart muscle due to diseases that affect the muscle itself, rather than being mediated through heart attacks from coronary artery disease. Included among the cardiomyopathies are subgroups of dilated and hypertrophic cardiomyopathy, with the dilated form being far more common. This group of causes collectively accounts for 10-15% of all SCDs. All of the other heart diseases combined account for no more than 5-10% of all SCDs. These specific statistics apply to the general population, but are dominated by population subsets over the age of 35 years, as the incidence of coronary heart disease and SCD increase rapidly with increasing age.[3]

For younger subsets of the population, uncommon or rare heart disorders are the most common causes of SCD, in contrast to common diseases such as coronary artery disease and cardiomyopathies that dominate in the middle-aged and older adult population. Recent data has demonstrated an association between left ventricular hypertrophy (most common due to inadequately treated high blood pressure) or unexplained fibrosis (scarring) of the heart muscle and the risk of cardiac arrest. The rare disorders include a number of inherited, developmental, and acquired diseases. The available literature suggests that myocarditis is a major contributor to SCD in this age group. The relative frequency, compared to other causes, is uncertain because of inconsistent diagnostic criteria in the older studies. Finally, since the majority of cases of myocarditis are mild or benign, the chances of an affected individual dying suddenly are small, especially after the acute phase has healed. In addition, many "rare syndromes" that are genetically determined and inherited among families, contribute to deaths in this age group. These include hypertrophic cardiomyopathy (HCM) as one of the more common causes, in addition to right ventricular dysplasia, the long QT interval syndromes, Brugada syndrome, congenital abnormalities of the coronary arteries or heart valves, and a

number of poorly-defined structural causes that are only now beginning to be understood.[5]

When no explanation for ventricular fibrillation or pulseless ventricular tachycardia (i.e., tachyarrhythmic or shockable cardiac arrest as the mechanism for sudden death) is identified, the event is usually labeled "idiopathic ventricular fibrillation." As diagnostic capabilities and clinical insight have improved in recent years, the number of deaths so classified has decreased. A small residual (likely less than 1 percent) still exists, but it is likely that the magnitude of this classification will continue to contract over time.

The final category of SCA includes those events in which an exogenous factor is responsible for a fatal heart rhythm disturbance. This group includes physical causes (e.g., *commotio cordis*) resulting from blunt trauma to the front of the chest by objects such as hockey pucks and baseballs,[6] electric shocks triggering fatal arrhythmias, and certain other forms of chest trauma, including restraint of breathing. In regard to the latter, excessive or improper restraint (especially in the prone position) may cause inadequate respiratory excursions, leading to respiratory failure and cardiorespiratory arrest.

Exogenous factors also include chemical substances. Among the latter, cocaine abuse is among the more common of such causes in the United States. Acute excess alcohol consumption, especially binge drinking with very high blood alcohol levels, has been associated with a limited risk of SCD, especially in the presence of pre-existing heart disorders.

The entity referred to as "acquired long QT interval (LQT) syndrome" is another example of exogenous factors. Historically, this has been linked to certain categories of drugs, such as the antiarrhythmic drugs, among others. In recent years, it has been demonstrated that a substantial subset of victims of "acquired" LQT are individuals who are susceptible to this response based on their genetic profiles. Such victims may have normal EKGs in the absence of the offending agents, and therefore are unrecognizable short of either genetic studies or exposures to drugs that allow expression of the syndrome. The frequency of such mutations or functional polymorphisms among the general population is currently unknown, but the incidence of proarrhythmic responses to potentially offending substances is thought to be less than 1-3%. The figure is lower in normal subjects and likely higher among those with advanced heart disease.

III. Onset of Cardiac Arrest: Predisposing Factors, Prediction, and Triggers

SCD is commonly initiated by interactions between pre-existing structural heart disease and transient physiological factors that alter the state of the heart at the time of onset of the cardiac arrest. Under such circumstances, the underlying substrate constitutes an anatomic or molecular substrate that establishes a predisposition to cardiac arrest, and the transient trigger is a significant contributing factor without which the cardiac arrest would not have occurred at a specific point in time. Among the structural predisposing factors, coronary atherosclerosis and the non-ischemic cardiomyopathies (primary heart muscle disease) account for 90–95 percent of SCDs in middle age or older subjects. In the younger population, the substrate is more likely to be an enlarged heart from high blood pressure, scarring of the heart muscle from prior viral infections, or other causes. A general example of this interaction comes from the Northridge earthquake in California in 1994. On the day of the earthquake, the incidence of SCA was 4 to 5 times higher than had been observed historically during the week before that event or during the same week going back 3 or 4 years. The conclusion was that the earthquake served as a stressful triggering event that initiated cardiac arrest in individuals who are susceptible to such events and brought them together at one point in time. This general principle can be applied to random individual stressors as well, raising the question whether an intensely stressful situation can be the initiator of a cardiac arrest in an individual with or without disease, but not at high risk for a cardiac arrest at that point in time.

The various structural disorders may interact with transient factors, such as acute interruption of blood flow (ischemia) in the case of coronary artery disease, changes in ventricular pumping function (heart failure) in both ischemic- and non-ischemic cardiomyopathies, electrolyte shifts, and autonomic (e.g., adrenalin) fluctuations.[7] However, transient triggering factors may not be identifiable in many individual cases.

Attributing coronary artery disease as the cause of a specific SCD is straightforward when post-mortem examination reveals a fresh blood clot occluding a coronary artery, with or without evidence of heart muscle injury or necrosis (cell death), or the presence of severe coronary artery disease (e.g., cholesterol plaques) in the absence of any other identifiable cause of SCD. However, it is more problematic in the circumstance of uncertainty about coronary artery disease status, especially in the presence of strong risk factors. The fundamental problem is that fact that transition from stable coronary artery plaques to an unstable state, initiating the formation of a hemorrhage into a plaque or a blood clot that occludes a coronary artery, is the most common mechanism of triggering a cardiac arrest and SCD due to CHD. This sequence can occur even when the underlying coronary artery abnormality appears minimal on a coronary angiogram, even days or weeks prior to the cardiac arrest. Finally, SCA can be initiated by spasm of a coronary artery. This generally occurs in the presence of a plaque that is not sufficient to trigger an event on its own, but makes the blood vessel susceptible to closing off when it goes into spasm.

IV. Analysis of the Events Surrounding the Cardiac Arrest in Andrew Layton

Andrew Layton was a 26-year-old male who died on January 5, 2013, as a consequence of a cardiac arrest that occurred on January 1, 2013. Based upon the various reports, videos, and medical records available for my review, I am able to determine the following sequence of events:

1. Mr. Layton had been initially noticed as having errant behavior and ultimately falling asleep in a Hy-Vee Convenience Store in the early morning hours of January 1, 2013, as a consequence of apparent heavy alcohol intake late in the evening of December 31, 2012 or early morning hours of January 1, 2013.

2. When the police were called they woke him and when he tried to walk away, they subsequently took him to the floor, restrained him, and he became agitated and combative. The form of restraint used was handcuffing his hands behind his back, tying his ankles together, and deploying a hobble-strap on his ankles and "hog-tying" him by flexing his legs posteriorly and tying the hobble strap from his ankles to his wrists, all of this while maintaining him in a prone position, and applying pressure to his back and legs as they considered necessary. A spit hood was also deployed, which remained in place until Mr. Layton arrived at the Blue Earth County jail in cardiac arrest (see below). After the police applied the prone restraints, an ambulance was called by them to transport him to the local jail, since the police responders believed that his level of consciousness was sufficient to conclude that he did not require hospitalization for a life-threatening drug or alcohol toxicity state. Based on videos and deposition testimony, Mr. Layton was hog-tied in the prone restraint position from approximately 4:54 AM until shortly after the ambulance arrived at the Hy-Vee at 5:05 AM. During transport to the jail, the ambulance was manned by two paramedics (Burt and Drews), and two police officers (Best and Baker), who also rode in the ambulance.

3. When Mr. Layton was placed in the ambulance, his legs were released from the "hog-tie" position, but remained restrained by tying his ankles to the stretcher, and he remained handcuffed with his hands behind his back. On the ambulance transport stretcher, Mr. Layton was tied down in the prone position with one stretcher strap across his lower legs, another strap over his lower back, and a third strap across his upper/mid-back. It is noteworthy that the types of straps used are standard on ambulance stretchers used to transport patients within hospitals or in transport vehicles. The patients are usually in the supine position with the straps tight to avoid falling, but patients should not be transported on a stretcher

in a restrained prone position because of the potential for limiting the patient's breathing movements. The same stretcher restraint positions in persons in the supine position would be much less likely to create a risk for breathing complications. In addition to being strapped down in a prone position, a pillow was placed under Mr. Layton's head and left shoulder area. This further restricted chest movement by angling Mr. Layton's neck and upper chest. There is testimony that Officer Baker would push Mr. Layton's back down or hold his head and shoulder down with force anytime Layton would fight against the restraints. Thus, the transport from the Hy-Vee to jail was carried out with Mr. Layton in a restrained prone position, with little ability to adjust his position in response to his breathing status, and a police officer countering any of his movements with downward force. These circumstances and actions would neutralize any natural breathing reflexes that were triggered by breathing impairment as a consequence of prone restraint.

Based upon the information available in the Gold Cross Ambulance log (Run # 69), and the depositions of the 2 paramedics, Mr. Layton had remained in prone restraints during the entire transport from the scene of the confrontation at the Hy-Vee Store to his arrival at the jail. The ambulance paramedics stated that Mr. Layton had a pulse and was breathing during transport. That determination was based upon palpating his radial pulse and feeling the back of his chest for respiratory movements. In addition, however, they also reported that when they performed an initial assessment upon arrival at the Hy-Vee, Layton was tachypneic at that time and he continued to be tachypneic during transport to the jail. Tachypnea is a rapid pattern of breathing that is a reflex action to impaired air exchange. Tachypnea is a recognizable clue to inadequate breathing under the circumstances of this case; and the paramedics and police officers knew, or should have known from their training, to avoid excessive prone restraint, except for a very short periods of time, such as to place handcuffs on the person. In addition, he was unintelligible and incoherent.

4. In their depositions, the paramedics also stated that they were not able to place a pulse-oximeter on his finger because it slipped off, and they do not produce a log of Mr. Layton's heart rate, breathing rate or depth, nor did they monitor his blood pressure during transport. It is also stated in the ambulance log that he became "quiet" shortly before arriving at the jail and appears to have gone into cardiac arrest immediately upon arrival at the jail, or just before or after arrival.

5. The time log from the ambulance report states an arrival time at the jail of approximately 5:21 AM on January 1, 2013. The records also reflect

that the AED available in the ambulance was not deployed until 5:26 AM. When the AED was deployed, the initial rhythm recorded was analyzed as a non-shockable rhythm ("no shock advised" on print out) and CPR was continued, in addition to intubation and administration of epinephrine. The log from Run #70 lacks some clarity regarding the life support activities. According to the Gold Cross log for Run #70, at 5:22 AM one of the responders (Burt) was performing chest compressions and the other responder (Drews) was addressing respiratory issues, initially with a BVM and subsequently with insertion of an oropharyngeal airway at 5:22 AM. It should be noted that the time of initiation of chest compressions recorded in the ambulance log is different than the time on the video from the jail – the latter shows a start time at 5:25 AM – a 3 minute discrepancy. Continuing with the log data, however, at 5:23 AM Burt was starting an IV, and then continued to start infusion through the IV (5:25 AM) and Drews was putting in an ETT. After this, Burt is administering an epinephrine infusion at 5:27 AM, and subsequently at 5:31 AM giving vasopressin. The first shockable rhythm was identified at 5:34 AM and a shock was delivered by the AED at that time but did not restore a spontaneous rhythm; a second shock as delivered at 5:36 AM and it restored an organized rhythm with a pulse and blood pressure, but Layton did not regain consciousness. Therefore, Layton's minimum down-time without a spontaneous circulation was 13 minutes, assuming the SCA occurred at 5:21 AM. It would have been longer if the cardiac arrest had occurred prior to arrival at the jail. Once he had a return of circulation, Mr. Layton was transported to Mankato Hospital.

6. Mr. Layton arrived at Mankato Hospital at 5:52 AM, and he was evaluated by a physician at 5:55 AM. He was found to remain unresponsive in the presence of an adequate pulse and blood pressure, and received prompt therapeutic hypothermia (TH). His blood alcohol level was 143 mg/dL on admission. To put this in perspective, most states define the DUI level in the range of 80-100 mg/dL, and fatal levels are in the range of 300-400 mg/dL, with significant degrees of alcohol-associated mental and physical impairment occurring in the range of 200-250 mg/dL. When considering the time from the SCA to arrival at Mankato Hospital, it is reasonable to assume that his blood alcohol level was under 200 mg/dL at the time of the SCA. Urine toxicology screen was positive for amphetamines and THC, but no quantitation is available.

7. After completing the TH protocol, Mr. Layton remained unresponsive and non-communicative, suggesting that the anoxic brain damage from the SCA was more-likely-than-not going to be irreversible. He never regained consciousness subsequent to that, even after withdrawal of sedation. He died four days later, on January 5, 2013.

8. The Death Certificate signed by Dr. Eric Evans cites atherosclerotic coronary artery disease as the immediate cause of death, while the post-mortem report by the Ramsey County Medical Examiner cites acute pneumonia due to probable excited delirium as the primary cause of death, also citing a background of cardiorespiratory arrest following struggle (physical exertion) with law enforcement on 1-1-13. In fact, both pneumonia and coronary atherosclerosis with a 75% occlusion of the left anterior descending coronary artery were found at autopsy, but neither of these is the actual cause of death (see "Opinions"). In addition, he had marked left ventricular hypertrophy and an abnormally thick LV wall, both likely secondary to documented high blood pressure, which had been untreated due to non-compliance.

V. Opinions on Causation and Standard of Care

Among the questions that I have been asked to evaluate in this case are the cause of Mr. Layton's cardiac arrest, the question whether the cardiac arrest could have been prevented, and whether a better outcome could have been achieved in light of the response to the cardiac arrest by the Gold Cross Paramedics.

A. Cause of cardiac arrest and subsequent biological death:

1. Mr. Layton was found to have premature coronary atherosclerosis at post-mortem examination, manifest as an estimated 75% occlusion of the left anterior descending coronary artery. This is quite unusual at his age, but not unheard of. However, it is well recognized that postmortem estimates often overestimate the degree of occlusion present during life when blood pressure in present in the vessel, and based upon the histology that I observed, it is my opinion that the occlusion during life was less than 75%. Moreover, there was no evidence histologically of plaque inflammation, disruption, platelet aggregation, or thrombosis factors, which closely associate with SCA due to coronary artery plaques. Coronary artery spasm superimposed on the plaque is another possibility for triggering a cardiac arrest. Based upon the histological evidence, I do not believe that the coronary atherosclerosis was the cause of the cardiac arrest within a reasonable degree of medical probability, but cannot exclude coronary artery spasm triggered by the environmental circumstances.

2. Pneumonia was present at the time of Mr. Layton's death. However, pneumonia is a known complication of cardiac arrest with persistent coma, and very commonly seen at the time of death in post-cardiac

arrest comatose patients. As such, it is not viewed as an independent cause of death. Rather, it is considered a complication of the sequence of cardiac arrest>coma, and contributes to the timing and mechanism of biological death, but the death itself is a result of SCA followed by coma, without which the pneumonia would not have occurred. Based upon reliable medical information in peer reviewed publications, it can be stated within a reasonable degree of medical probability that a post-cardiac arrest victim with the level of brain damage observed in Mr. Layton will not survive to hospital discharge, in the presence or absence of pneumonia.[8,9]

3. Linking the pneumonia to so-called excited delirium has no basis. First, excited delirium is not recognized as a true medical entity, and secondly, cardiac arrest is commonly associated with aspiration during resuscitation that creates the nidus for pneumonia in patients who remain comatose. Therefore, my opinion, to a reasonable degree of medical probability, is that pneumonia was, more-likely-than-not, a result of aspiration during the onset of cardiac arrest or soon after resuscitation, or a complication of the comatose post-SCA state, and not a primary cause of death.

4. Another consideration of potential causes of Mr. Layton's cardiac arrest is mechanical asphyxia secondary to impaired breathing from improper or excessive restraints. Based on the testimony regarding what happened during the pre-ambulance prone restraint by the police officers and the ambulance log of the 8-minute ambulance trip from Hy-Vee to the jail, a respiratory complication secondary to the nature of the prone restraint is the most likely probability of causation. The nature of the forceful prone restraint, Layton's described breathing pattern of tachypnea, and the initial electrical mechanism recorded during the cardiac arrest, namely pulseless electrical activity (PEA), all come together to make it most likely, within a reasonable degree of medical probability, that Layton suffered a cardiorespiratory arrest secondary to mechanical impairment of breathing. PEA is the mechanism that commonly occurs in cardiac arrest triggered by respiratory problems. [10]

B. <u>Standard of care in responding to a cardiac arrest</u>:

1. After noting that Mr. Layton was tachypneic while in forcible prone restraint, the police officers and paramedics failed to take action to reposition Layton, in order to improve his breathing status. Had they done so, it is likely that Mr. Layton would have resumed normal air exchange and avoided the cardiorespiratory arrest.

2. Another issue relates to the deployment of the AED. It appears that the cardiac arrest was suspected or recognized at approximately 5:21-5:22 AM, and the AED was not deployed until 5:26 AM. The standard for response to a cardiac arrest calls for immediate institution of chest compressions upon identifying a cardiac arrest and simultaneously contacting emergency rescue (911). The latter was not relevant in this case because paramedic responders were already on-scene. The AED is then sought and brought to the patient's side. In usual circumstances, this can consume considerable time, but in this case the responders were in an AED-equipped ambulance and were fully trained in how to deploy the AED. Therefore, the AED should have been operational within one minute or less. The fact that this does not appear to have occurred might be a significant contributing factor to the ultimate outcome. If Mr. Layton indeed had a shockable rhythm at the onset of the cardiac arrest, the risk for significant brain damage could have been greatly reduced or avoided if the AED had been deployed immediately. This assumes that the responders accurately reported that Mr. Layton was conscious up to 1 minute or less prior to arrival at the jail. If so, and if an initial shockable rhythm was present, the opportunity for CNS preservation more-likely-than-not was lost because of delayed AED deployment.

3. The report on Run #70 by the Gold Cross Ambulance and the video from the jail raise serious questions about whether the life support activities by the Gold Cross Ambulance paramedics were done properly. There appears to be a delay of at least 3-4 minutes between the time that it was reasonable to suspect a cardiac arrest and the initiation of chest compressions by paramedic Burt. This assumes that the cardiac arrest did not occur in the ambulance prior to arrival at the jail. With a 2-person response team, one individual is expected to provide continuous chest compressions immediately upon identifying a cardiac arrest, interrupting only for evaluating the rhythm once the AED is deployed. If the rhythm is non-shockable, there should be 2 minute cycles of continuous CPR followed by a brief pause to evaluate the rhythm and pulse, and then resumed for another 2 minutes. The data available suggest much more frequent interruptions, generally of the proper duration, but not the continuous 2 minute cycles of continuous compressions between pauses. The second responder is expected to manage respiratory factors and other activities. According to the Gold Cross log, at 5:22 AM one of the responders (Burt) was performing chest compressions (note discrepancy with video time of 5:25 AM for start of chest compressions) and the other responder (Drews) was addressing respiratory issues, initially with a BVM, and subsequently inserting an oropharyngeal airway at 5:22 AM, and at 5:23 AM Burt was starting an ID. Burt then continued to start an

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infusion through the IV and at this point (5:25 AM) Drews was putting in an ETT. After this Burt is giving an epinephrine infusion and subsequently at 5:31 AM vasopressin. During this period, one of the police officers was providing chest compressions. The guidelines for BLS and ACLS clearly state that chest compressions is the single most important component of CPR and should be continued in 2 minute cycles with minimal interruptions. Therefore, there seems to be deviations from this protocol during the response to the cardiac arrest at least up to the point in time when the first shock was delivered at 5:34 AM.

My foregoing opinions regarding standard of care are based on the currently available facts of the case, and of causation are based upon a reasonable degree of medical probability, in consideration of the facts of the case, in conjunction with my personal education, background, knowledge, and experience, and reliable medical information published in peer-reviewed journals. I reserve the right to expand or alter any of my opinions based upon any new or additional information provided to me in the future.

Notary Public State of Florida
Stephanie Ogando
My Commission FF 976910
Expires 03/30/2020

Robert J. Myerburg, M.D.
Miami, Florida

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8. 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science. Circulation, 122 (Supplement 3):S640, 2010.

9. IOM (Institute of Medicine) 2015. Committee on the Treatment of Cardiac Arrest: Current Status and Future Directions: Strategies to improve cardiac arrest survival: A time to act. Washington, DC: The National Academies Press.

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May 31, 2016

PERSONAL

2. **Robert J. Myerburg, M.D.**

3. **Home Phone:**

4. **Office Phone:** (305) 585-5523 **Fax:** (305) 585-7085

5. **Home Address:** 11300 S.W. 94th Avenue
Miami, Florida 33176-4200

6. **Current Academic Rank:** Professor, Department of Medicine
Professor, Department of Physiology

6a. **Current Track of Appointment:** Tenure

7. **Primary Department:** Department of Medicine, Division of Cardiology

8. **Secondary Appointments:** Department of Physiology

9. **Citizenship:** U.S.A.

10. **Visa Type:** N/A

HIGHER EDUCATION

11. **Institutional:**

Johns Hopkins University Biological Sciences		September, 1954 - June, 1957
University of Maryland School of Medicine	M.D.	September, 1957 - June, 1961

12. **Non-Institutional:** N/A

13. **Certification, licensures, boards (* currently active):**

Maryland State Board of Medical Examiners #053760	1961
Certified by National Board of Medical Examiners	1962
Louisiana State Board of Medical Examiners #2284R	1964
New York State Department of Education #103549	1969
*Florida State Board of Medical Examiners #15305	1970
Diplomate, Am Board of Internal Medicine #27950	1968
Diplomate, Subspecialty Board of Cardiovascular Diseases, Am Board of Internal Medicine #27950	1970
Certificate of Special Competence, Clinical Cardiac Electrophysiology, Am Board of Internal Medicine #27950	1998

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EXPERIENCE

14. Academic:

University of Miami School of Medicine	Assistant Professor of Medicine	Jan. 1, 1970 - May 31, 1972
University of Miami School of Medicine	Assistant Professor of Physiology	June 1, 197 - May 31, 1972
University of Miami School of Medicine	Associate Professor of Medicine & Physiology	June 1, 1972-May 31, 1974
Miami Veterans Administration Medical Center	Chief, Cardiology Section, Medical Service	Jan. 1, 1970-Aug. 31, 1974
University of Miami School of Medicine	Director, Division of Cardiology	September 1, 1973-Sept. 1, 2004
University of Miami School of Medicine	Director, Cardiology Training Program	September 1, 1973-January 1, 2008
University of Miami School of Medicine	Professor of Medicine & Physiology	June 1, 1974 - Present

15. Hospital Appointments:

Jackson Memorial Hospital	1970 to present
VA Medical Center	1970 to present
University of Miami Hospital and Clinics	1970 to present
Baptist Hospital	1995 to 2009
Cedars Hospital	2002 to 2007
University of Miami Hospital	2007 to present

16. Non-Academic: N/A

17. Military: U. S. Public Health Service Heart Disease Control Program — July 1, 1962-June 30, 1964

PUBLICATIONS

18. Books, chapters in books, and monographs published:

1. Hurst JW, Myerburg RJ. **Introduction to Electrocardiography**. New York: McGraw-Hill Book Co. 1968: 314.

2. Myerburg RJ. Sudden death. In: Hurst JW, Logue RB, eds. **The Heart**. 2nd edition. New York: McGraw-Hill Book Co. 1970:551-555.

3. Marriott HJL, Myerburg RJ. Recognition and treatment of cardiac arrhythmias and conduction

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disturbances. In: Hurst JW, Logue RB, eds. **The Heart**. 2nd edition. New York: McGraw-Hill Book Co. 1970:489-536.

4. Myerburg RJ. Electrocardiographic diagnosis of sinus node rhythm variations and S-A block. In: Schlant RC, Hurst JW, eds. **Advances in Electrocardiography**. New York: Grune & Stratton. 1972:73-80.

5. Myerburg RJ, Gelband H, Hoffman BF. Electrophysiologic function of the distal A-V conducting system and comments on its clinical relevance. In: Schlant RC, Hurst JW, eds. **Advances in Electrocardiography**. New York: Grune & Stratton. 1972:129-144.

6. Myerburg RJ, Nilsson K, Castellanos A, Gelband H. The mechanism of ventricular endocardial excitation. In: Dreifus LS, Likoff W, eds. **Cardiac Arrhythmias**. New York: Grune & Stratton. 1973:181-193.

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8. Castellanos A, Agha AS, Castillo CA, Myerburg RJ. Ventricular activation in the presence of Wolff-Parkinson-White syndrome. In: Dreifus LS, Likoff W, eds. **Cardiac Arrhythmias**. New York: Grune & Stratton. 1973:451-465.

9. Hurst JW, Myerburg RJ. **Introduction to Electrocardiography**. 2nd edition. New York: McGraw-Hill Book Co. 1973, 319 pp.

10. Myerburg RJ. Sudden death. In: Hurst JW, ed. **The Heart**. 3rd edition. New York: McGraw-Hill Book Co. 1974:585-591.

11. Marriott HJL, Myerburg RJ. Recognition and treatment of cardiac arrhythmias and conduction disturbances. In: Hurst JW, ed. **The Heart**. 3rd edition. New York: McGraw-Hill Book Co. 1974:502-569.

12. Myerburg RJ, Nilsson K, Castellanos A, Lazzara R, Befeler B, Gelband H. The intraventricular conduction system and patterns of endocardial excitation. In: Cigueira Gomes, eds. **Recent Advances in Ventricular Conduction**. Adv. Cardiol., Basel, Switzerland: S. Karger Publishing. 1975; 14:2-14.

13. Myerburg RJ, Castellanos A, Bassett AL, Gelband H. Functional properties of the distal A-V conducting system. In: Cigueira Gomes, eds. **Recent advances in Ventricular Conduction**. Basel, Switzerland: S. Karger Publishing. 1975; 14:94-104.

13a. Castellanos A, Levy S, Befeler B, Myerburg RJ. Mechanisms determining the ventricular rate in Wolff-Parkinson-White arrhythmias: In: Cigueira Gomes, eds. **Recent Advances in Ventricular Conduction**. Basel, Switzerland: S. Karger Publishing 1975; 14:221-232.

14. Castellanos A, Befeler B, Agha AS, Myerburg RJ. Intracardiac electrocardiography in evaluation of the positive QRS complexes in lead V1 elicited by right ventricular apical stimulation, Ch. 13. In: Narula OS, ed. **His bundle Electrocardiography and Clinical Electrophysiology**. Philadelphia: F.A. Davis. 1975:261-271.

15. Gottlieb S, Sheps DS, Myerburg RJ, Miale A: Applications of diagnostic ultrasound and radionuclides to cardiovascular diagnosis. Part I. Acquired cardiovascular diseases in the adult. In: Freeman LM, Blaufax

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M, eds. **Nuclear Medicine and Ultrasound**. New York: Grune & Stratton. 1975:67-100.

16. Myerburg RJ, El-Sherif N. Electrocardiographic diagnosis of sinus rhythm variations and S-A block, Ch. 4. In: Schlant RC, Hurst JW, eds. **Advances in Electrocardiography**. 2nd edition. New York:Grune & Stratton. 1976:59-70.

17. Castellanos A, Lemberg L, Sung RJ, Myerburg RJ. Dynamic vectorcardiography in the evaluation of left hemiblocks. In: Hoffman & Hamby, eds. **Vectorcardiography**. Amsterdam: North Holland Publishing Co. 1976:99-111.

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19. Gelband H, Nilsson K, Sung RJ, Bassett AL, Myerburg RJ. Electrophysiology of the intact neonatal canine atrioventricular conducting system. In: Wellens HJJ, ed. **Conduction System of the Heart**. Leiden, The Netherlands'. E. Stenfert Kroese B.V, and Philadelphia: Lea & Febiger. 1976.

20. Myerburg RJ, Gelband H, Castellanos A, Nilsson K, Sung RJ, Bassett AL. Electrophysiology of endocardial intraventricular conduction: The role and function of the specialized conducting system. In: Wellens HJJ, Lie KI, Janse MJ, eds. **The Conduction System of the Heart**. Philadelphia: Lea & Febiger. 1976:336-359.

21. Castellanos A, Myerburg RJ. **The Hemiblocks in Myocardial Infarction**. New York: Appleton-Century-Crofts. 1976.

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24. Castellanos A, Befeler B, Agha AS, Myerburg RJ. Contribution of catheter electrodes to study of intraventricular conduction in the human heart; with special reference to bundle branch block, ventricular pacing, and Wolff-Parkinson-White syndrome, Ch. 8. In: Schlant RC, Hurst JW, eds. **Advances in Electrocardiography**. 2nd edition. New York: Grune & Stratton. 1976:143-169.

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28. Hurst JW, Myerburg RJ. The Electrocardiogram. In: Walker HK, Hall WD, Hurst JW, eds. **Clinical Methods: The History, Physical and Laboratory Examinations**. Butterworths, Woburn, Mass. Chapter 10. 1977:945-1030.

29. Myerburg RJ, Gelband H, Bassett AL. Physiology of the ventricular specialized conduction system. In: Roberts NK, Gelband H, eds. **Cardiac Arrhythmias in the Neonate Infant and Child**. New York: Appleton-Century-Crofts. 1977:55-90.

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41. Sheps DS, Myerburg RJ. Drug interactions involving antiarrhythmic drugs, Ch. 4. In: Petrie JC, ed. **Cardiovascular and respiratory disease therapy**. Volume 1. The Netherlands: Elsevier/North Holland

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